Foodborne Viruses: Their Importance and Need for Research[1]

ABSTRACT

All viruses known to be normally transmissible through foods and of concern to human health emanate from the human intestine. The outbreaks of hepatitis A and recently of gastroenteritis attributed to Norwalk-like viruses most likely developed from feces-contaminated fingers of infected food handlers or water polluted with feces. With few exceptions no recorded outbreak has depended on the ability of virus to withstand even limited heating in food. New and better methods of detection are needed for hepatitis A and Norwalk viruses in foods. It has been well documented that international trade in food products of animal origin can result in the introduction of animal disease into areas in which the disease does not exist. This fact has given rise to programs of research and development for industrially applicable technology to rid animal products from the agents of animal diseases. The survival of viruses inclusive of etiological agents of foot-and-mouth disease, African swine fever, swine vesicular disease and hog cholera virus is reviewed in this paper and new research approaches are suggested. The general need for additional research of foodborne viruses is discussed.

Epidemiological, clinical, and laboratory investigations indicate that viral and rickettsial diseases can potentially be transmitted via foods and feeds. An accurate assessment of foods acting as vehicles for such diseases has not been completed, and the relative food safety significance of foodborne viruses and rickettsia is largely unknown. Development of new serological methods for detection of viruses and development of better procedures for extracting viruses from food samples makes it appropriate to consider such an assessment now. An accurate assessment of the relative importance of foodborne viruses will require greater knowledge of: (*a*) mechanisms of transmission; (*b*) minimal infective doses for man or other animals; (*c*) sources of contamination; (*d*) host ranges and ability of animal viruses to infect humans; and (*e*) the ability of viruses to withstand environmental, chemical, or physical inactivation. The resources which can be brought to bear to obtain this knowledge are limited, requiring that the possible roles of government, academic and industrial groups be defined to avoid duplications of effort.

FOOD CONTAMINATION BY HUMAN ENTERIC VIRUSES

All viruses known to be transmissible through foods and of concern to human health in the United States emanate from the human intestine. Four "kinds" of viruses, all enteric, have been transmitted via foods in the U.S.: hepatitis A, Norwalk-like agents, polio viruses (not since 1949), and echovirus 4 (*18*). Many outbreaks of hepatitis A and, more recently, a few of gastroenteritis attributed to the Norwalk-like viruses have been reported (*18*). *Coxiella burnetti* (not a virus) and tick-borne encephalitis viruses which can be trasmitted in unpasteurized milk are not now transmitted via foods in North America and are excluded by definition. Enteroviruses other than those already named, as well as non-A, non-B hepatitis viruses, rotaviruses and other gastroenteritis viruses, are surprisingly absent from reports of foodborne disease in the United States (*18*).

The proximate source of the contaminating viruses (based largely on the hepatitis A record) is either the feces-contaminated fingers of an infected food handler or water polluted with feces, though the possibility of insects serving as mechanical vectors for fecal viruses cannot be excluded entirely (*18*). Any kind of food is at risk of hands-on contamination. Such contamination is preventable only by very careful personal hygiene and the

---

[1]*This paper is adapted from a symposium presented at the Annual Meeting of the American Society for Microbiology, March 8, 1984.*

[2]*Foreign Animal Disease Diagnostic Laboratory APHIS, USDA, P.O. Box 848, Greenport, Long Island, New York 11944.*

[3]*Food Research Institute (Department of Food Microbiology and Toxicology), World Health Organization Collaborating Centre on Food Virology, and Department of Bacteriology, University of Wisconsin, Madison, WI 53706.*

[4]*Department of Veterinary Public Health, Texas A&M University, College Station, TX 77843.*

[5]*Virology Branch, Division of Microbiology, Center for Food Safety and Applied Nutrition, Food and Drug Administration, 1090 Tusculum Avenue, Cincinnati, OH 45226.*

[6]*Plum Island Animal Disease Center, USDA-ARS-NAA-PIADC, P.O. Box 848, Greenport, Long Island, NY 11944.*

[7]*Food Safety Laboratory, Eastern Regional Research Center, ARS, USDA, 600 E. Mermaid Lane, Philadelphia, PA 19118.*

prevention of food handling by infected individuals. Since symptoms of hepatitis A, at least, may not be manifest at the time that contamination occurs, preventing infected food handlers from contacting food is difficult. Water contaminated by feces most often affects shellfish, but watercress (*51*) and cold cuts (*50*) have been affected. Such contamination is preventable by not using water for feces disposal or by treating water and wastewater more effectively. An instance of contamination via a fomes occurred when cafeteria trays with which food was in contact were contaminated by an infected worker (*27*). Vectors (e.g., flies and roaches) and aerosals have not been firmly implicated as sources of contamination. The point of contamination is most often during the final preparation and serving of the food (foodservice or in the home). It is seldom in food processing, storage, or distribution, unless one considers the preparation and spreading of icings on baked goods to be processing. Shellfish are different in that they may accumulate viruses and are usually contaminated before harvesting, but this is true of practically no other food.

The lack of stability of viruses in foods may be the factor that has prevented transmission of some viruses via foods. However, with few exceptions, principally involving shellfish contaminated with hepatitis A or the Norwalk agent which are especially heat resistant, no documented outbreak depended on the ability of viruses to withstand even limited heating in food.

Detection methods for viruses in foods are primarily of value for verification that a food was the source of virus in an outbreak rather than to monitor foods for presence of viruses because of the expense, relative lack of sensitivity, and the time consumption of such methods. Increasingly quick, efficient, sensitive, inexpensive cell culture methods are being developed for detecting enteroviruses, and perhaps rotaviruses, in shellfish and other foods (*18,19*). Such methods will be of use if it can be shown that these viruses are good indicators of the presence of hepatitis A virus and the Norwalk-like viruses in the foods tested, but they will not detect either of the last-named agents in food, and neither will any other currently available method. Neither the hepatitis A virus nor the Norwalk agents will replicate in cell cultures in a way that is useful in detecting the viruses in food samples, so the best hope is that methods [e.g., radioimmunoassay (RIA), enzyme-linked immunosorbent assay (ELISA), immune electron microscopy (IEM), and fluorescent antibody (FA) microscopy] for detecting these viruses as antigens in foods will be forthcoming, even though there is little prospect that these will rival cell culture methods in sensitivity.

Of hepatitis A reported in the United States during 1977-1981 (the most recent 5 years for which year-end summaries from the U.S. Centers for Disease Control are available), 690 illnesses (or 0.47%) of the total of 145,949 were food-associated. It is unknown if foodborne hepatitis A is more seriously underreported than the disease as a whole in the United States. Of food-associated outbreaks of illness for the same 5 years, 63% of the reported outbreaks, comprising 47% of the outbreak-associated illnesses, were of undetermined etiology; 33 outbreaks of viral illness comprised 3.5% of the outbreaks of confirmed etiology and included 869 illnesses, or 2.6% of the outbreaks of known etiology. This may be contrasted with 6000 cases of gastroenteritis attributed to the Norwalk agent in two outbreaks in Minnesota in 1982; the implicated vehicles were butter-cream icing in one outbreak and salad in the other. There may be a great deal more foodborne viral illness occurring, if someone cares enough to look for it, but because the available methods lack the required sensitivity and rapidity the looking will have to be done epidemiologically, rather than by laboratory methods, for the time being.

DISSEMINATION OF VIRUS DISEASES OF ANIMALS THROUGH ANIMAL PRODUCTS

There are many documented incidents in which international trade in products derived from animals resulted in the introduction of foreign animal diseases into the domestic animal population (*1,8,37,44,45*). These outbreaks emphasize the need to regulate exports of animal products from nations in which economically devastating animal disease is endemic. There are many meat products and by-products on the international market today that originate in countries with animal diseases that do not exist in the United States. The viral diseases that we are most concerned with, and which will be discussed in this review are African swine fever (ASF; *15*), swine vesicular disease (SVD; *42*), hog cholera (HC; *40*), and foot-and-mouth disease (FMD; *41*).

The introduction of foreign animal disease via animal products can be very costly. The predicted cost of a wide-scale outbreak of FMD in the United States in the first year alone would be in excess of $4 billion in direct losses. Indirect losses have been estimated to be approximately 10 times greater (*38*). The swine industry in England was decimated by the introduction of SVD in 1972. An intensive eradication program cost $12 million in compensation fees alone in the first 5 years of the effort (*44*).

It has been claimed that the FMD outbreak of 1967-68 in the United Kingdom and the chronic focus of SVD in the pig population in England arose from imported pork products and meat scraps from diseased pigs (*8,44*). The same explanation has been proposed for the introduction of ASF into Portugal from the Portuguese African territories (*45*) and subsequently to Italy (*1*).

African swine fever is a highly contagious, often acute, viral disease of porcine animals characterized by fever, marked cyanosis of skin areas, and clearly apparent hemorrhages of the internal organs, particularly the lymph nodes, kidney and gastrointestinal mucosa. Mortality frequently approaches 100% in initial epizootics. ASF has been an important disease of swine in many parts of the world. Its clinical similarity to HC makes laboratory diagnosis critical. It has become a major concern to

the Western Hemisphere since its diagnosis in Cuba, Brazil, Dominican Republic, and Haiti (*1*).

Swine vesicular disease is a contagious viral disease of swine indistinguishable clinically from FMD, vesicular stomatitis, and vesicular exanthema of swine. Investigations of outbreaks incriminate the feeding of garbage contaminated with SVD virus-infected meat scraps as a major source of infection. The stability of the virus is such that it is not inactivated by the acid changes that occur in the musculature after death; thus the virus can be expected to withstand many of the processes used in product production. There is little or no decrease of infectivity in cold storage in uncooked pork or pork products.

Hog cholera is a highly infectious septicemia caused by a virus and characterized by generalized hemorrhages, 95 to 100% morbidity, and almost as high mortality. HC is present in most countries where swine are raised, with the exception of Canada, United States, Great Britain, and Australia.

Foot-and-mouth disease is an acute, highly communicable disease existing almost exclusively in domesticated and wild cloven-footed animals. The disease is characterized by formation of vesicles and erosions in the mucosa of the mouth and external nares (especially on the snout of pigs) and the skin between and above the hooves of the feet; other areas, including teats, may be involved. FMD virus is distributed throughout the body of the infected animal and can be found in different concentrations for various periods in the tissues, secretions, and excretions. Survival of the virus in the slaughtered animal is dependent on the stage of the disease at the time of death, on characteristics of the strain of virus, and on environmental factors such as temperature and hydrogen ion concentration. The virus of FMD in skeletal muscle is inactivated within 3 d after slaughter due to reduced pH. In contrast, the virus may survive for weeks or months in refrigerated internal organs, bone marrow, lymph and hemal nodes, glands, and residual blood (*21*).

Animal disease viruses vary in their persistence in animal tissues and secretions (Table 1). For example, FMD virus is inactivated within 48 h in skeletal muscle held at 4°C wherein pH drops below 6.0. However, in lymph nodes, coagulated blood, and bone marrow obtained from these same carcasses, the virus persists for a minimum of 120 d at 4°C (*20*). These latter tissues provide an environment that protects FMD virus from inactivation through decrease in pH associated with postmortem changes (*21*). High-titer virolactias have been detected in milk of lactating cows before the appearance of clinical FMD (*11,16*), and the virus has been detected in milk in the presence of antibody at 52 d after infection (*17*).

SVD virus is considerably more resistant to lower pH than FMD virus. SVD virus has been shown to persist for 11 months in swine carcasses held at 4°C (*24*). ASF virus persists for long periods in pork: 150 d at 4°C (*33*), 104 d at -4°C, and 188 d in frozen (-4°C) bone marrow (*9*). There are reports that rinderpest (RP) virus has survived in carcasses for 30 d and for 8 d in quarters that had been aged for 24 h at 4°C (*29*). RP virus has also been recovered from tissues 36 d after experimental infection and from carcasses buried for 2 months. Virolactias have been detected up to 45 d in recovered cows (*29*). The stability of HC virus in carcasses is comparable to that of RP virus, and it persists through 33 d in skin and 73 d in muscle (*2*).

EFFECTS OF FOOD PROCESSING ON FOODBORNE VIRUSES

Foods derived from animals are frequently processed by heating, lowering pH, salting, dehydration, irradiation, etc., to preserve and/or change the organoleptic quality of the food before serving. Processed foods are not necessarily free of viral agents of animal diseases (Table 2). Because of the practice of feeding garbage to livestock, virus survival in such products prepared from infected animals is a concern.

Food processing involving heating, controlled acid development, and irradiation can inactivate viruses. Mechanisms of virus inactivation include alteration of membrane protein and/or complexing or fragmentation of nucleic acid (*33*). Heating denatures capsid (viral surface) protein, thus preventing specific attachment of the virion to the cell receptor site, inactivating virion-associated enzymes needed for synthesis of progeny virus and interfering with removal of coat protein and release of viral nucleic acid. At temperatures above 70°C, unwinding of double-stranded viral nucleic acid, and random breaks in the sugar phosphate backbone are part of the irreversible changes that occur in the viruses (*33*). Extremes in pH also denature surface protein; and in addition, low pH prevents formation of templates for nucleic acid replication (*33*). Ionizing radiation involves nucleic acid strand fragmentation. During irradiation, strand scission occurs at 10 times the efficiency with single-stranded than with double-stranded viral nucleic acid (*33*). Baldelli et al. (*4*) found that a minimum of 17.5 kilogray (kGy) would inactivate FMD virus in blood, bone marrow and lymph nodes collected from infected pigs. McVicar et al. (*43*)

TABLE 1. *Persistence of foreign animal disease viruses in animal carcasses.*

Disease virus	Persistence (d) at 4°C	Tissue	Reference
Foot and mouth	120	Lymph nodes	Cottral (*20*)
Swine vesicular	330	Skeletal muscle	Dawe (*24*)
African swine fever	110	Skeletal muscle	Kowalenko (*34*)
Rinderpest	30	Skeletal muscle	Gillespie et al. (*29*)
Hog cholera	33	Skin	Anon. (*2*)

TABLE 2. *Survival of foreign animal disease viruses in products prepared from infected carcasses.*

Virus[a]	Product	Processing conditions	Effectiveness[b] of processing	Reference
FMD	Infected lymph nodes in ground beef	Retort cooking to 68.3°C	+	Heidelbaugh & Graves (*31*)
FMD	Ground beef	Cooked in nylon tubes to:		
		63°C	−	Blackwell et al. (*13*)
		79.4°C	+	Blackwell et al. (*13*)
FMD	Meatballs	Cooked in nylon tubes to:		
		93.3°C	+	Blackwell et al. (*13*)
SVD	Salami Sausage	Fermentation & curing	−	McKercher et al. (*39*)
ASF	Ham	Brining	−	McKercher et al. (*41*)
		Brining, retort cooking to 69°C	+	McKercher et al. (*41*)
HC	Ham	Retort cooking to 69°C	+	McKercher et al. (*41*)

[a]Foot-and-mouth disease (FMD), Swine vesicular disease (SVD), African swine fever (ASF), Hog Cholera (HC).
[b]+ = Virus not detected after processing; − = Virus detected after processing.

inactivated ASF virus in infected swine tissue with 20-kGy doses.

Most of the products mentioned in the following paragraphs were made from the carcasses of animals infected with the specific disease and sacrificed at the peak viremic phase. In general, the amount of virus in these products, prepared from selected carcasses, would be greater than would occur under more natural conditions. Processing included (*a*) heat, (*b*) drying and curing, and (*c*) salting and aging. The host animal was more sensitive than tissue culture for detecting residual viable viruses.

It would appear that 69°C is a critical temperature in the thermal processing of animal products because ASF, SVD, HC, and FMD viruses are inactivated at this temperature. ASF virus was not recovered from partially cooked canned hams heated to 69°C nor from ground lymph nodes from infected swine when the ground tissue was heated to 69°C. HC virus was not recovered from partially cooked canned hams heated to 69°C (*41*), which is in agreement with Stewart et al. (*47*) in whose studies a "flash" temperature of 71°C for 1 min inactivated HC virus in (2 cm^3) cubes of ham. Also FMD virus was inactivated in infected lymph nodes packed in ground beef when processed by retort cooking to an internal temperature of 68.3°C and in hams prepared from infected pigs when cooked to an internal temperature of 69°C (*30*). Heidelbaugh and Graves (*21*) found that the virus in lymph nodes from infected cattle was inactivated by heating to 69°C. McKercher et al. (*42*) had similar results with ground lymph nodes from infected swine. In contrast, Blackwell et al. (*13*) recovered FMD virus from lymph nodes of infected cattle after heating for 2 h at 69°C, 1 h at 80°C, and 0.25 h at 90°C. Further, the incorporation of 1% NaCl enhanced virus survival. In studies of meatballs cooked to an internal temperature of 93°C in nylon tubes was sufficient to ensure that the virus did not survive processing (*13*). In recent tube cooking trials at the USDA Plum Island Animal Disease Center, FMD virus in ground beef products survived a temperature of 72°C, but was inactivated in products cooked to an internal temperature of 79.4°C.

In dried pepperoni and salami sausages, ASF virus was recoverable throughout 15 d of the curing period. ASF virus was recovered in the muscle of salt-cured hams up to 5 months and in the bone marrow of these hams up to 6 months (*15*). Processed intestinal casings stored for 97 d caused clinical ASF and death when extracts were inoculated into pigs (*42*). Cured and dried pork products originating from countries where SVD is present are not permitted entry into the United States except for further-processing by heating to an internal temperature of 74°C (*49*).

Swine vesicular disease virus can survive for at least 400 d in dried salami and pepperoni sausages prepared by controlled acid fermentation (*39*). In processed intestinal casings, the virus persisted for at least 780 d nor was it inactivated by 24 h of exposure to 0.5% citric acid (*30*). SVD virus remains viable in the muscle, fat, and bone marrow of salt-cured hams for at least 6 months but is no longer viable after 10 months.

HC virus was recovered from dried pepperoni and salami sausages through 30 d of the curing period (*41*). However, processed intestinal casings produced clinical HC and death when extracts were inoculated into pigs after 147 d of storage (*42*).

Meat derived from animals infected with foot-and-mouth disease is not rendered free of the virus by the usual commercial procedures of ripening, boning, salting, and storage even if the muscle itself is free (*48*). Dhennin et al. (*26*) recovered FMD virus in sausages up to 56 d, in salted bacon for 190 d, and up to 183 d in ham fat (*26*). Residual FMD virus remained in processed intestinal casings from FMD infected pigs for as long as 250 d (*42*).

These studies all involved the use of carcasses of diseased animals, which is in itself a serious threat to the livestock industry. The risk of infecting livestock from human food products, such as the transmission of SVD to hogs via garbage, that could contain residual viruses is small. Nevertheless one must be aware that there is

a risk, however slight.

Table 3 summarizes the survival of FMD virus in dairy products. The FMD virus survived high temperature-short time (HTST) pasteurization at 72°C for 15 s in whole milk, skim milk, cream and pelleted cellular debris (*10,11*). Evaporation of whole milk samples after heating at 75°C for 3 min did not inactivate the virus (*32*). Viral infectivity was detected in milk after heating at 138°C for 2 s, but was not detected after ultra-high temperature (UHT) sterilization at 148°C for 3 s or longer (*23*). In addition, in milk from FMD-infected cows, the virus survived heating and in butter ripened with lactic acid cultures at pH 4.6 (*6*), in isoelectric precipitated hydrochloric acid casein at pH 4.6 (*22*), in cheese at pH 5.1 (*5*), and in sweet whey at pH 5.2 (*5,7*), but not those of acid whey, alpha-lactalbumin, beta-lactoglobulin, and lactose (*7*). In all heating trials, FMD virus present in milk as a result of infection was more stable than virus added to milk (*25*).

FMD virus on inanimate surfaces is readily inactivated by a variety of disinfectants at pH extremes of 5.8 or 11.0 (*46*). Acid formulations at a pH of 6.5 and below are routinely used in disinfecting procedures. Although infectious viral RNA survives heating at 100°C for 5 min, the virion is inactivated at subpasteurization temperatures (*3*).

In lymph node tissue, congealed blood, or bone marrow, FMD virus is more protected from disruption by heat and pH change than is virus in muscle. However, FMD virus, which persists for long periods of intestinal casings, is readily inactivated on exposure to 0.5% citric or lactic acid. This fact and the observation that FMD virus survived in casein but not in the acid whey by-product, suggested that protective complexes were being formed at the level of the secretory epithelial cell (*12*). In fact, such structures were observed by electronmicros-

Aging of meat stored in a chilling room at a temperature not below 2°C during 24 h is one of the requirements of the European Economic Community (EEC) for meat exporting countries affected with FMD. The objective of this regulation is to ensure that a pH below 6.0 has been reached before deboning, to guarantee inactivation of the FMD virus that might be present. Inactivation of FMD virus in meat from artificially infected animals has been studied in relation to the post mortem decrease of the pH in the muscle tissue. Studies have been made at two slaughter-houses that use standard commercial practices of electrical stimulation at low and high voltage (*28*). The post-mortem evolution of muscle pH was controlled in treated beef. It was concluded that the commercial practice of electrical stimulation reduces the time needed to obtain the pH values that guarantee the FMD virus inactivation in muscle, resulting in the possiblity of using electrical stimulation as an alternative method in the aging of meat to destroy FMD virus (*28*).

There is great potential and promise for development and application of food processing methods which can destroy the virus of animal diseases in foods. The develoment of such industrially practical techniques offers a relatively inexpensive way for technologically advanced nations to assist animal producing nations (especially in South America) to produce animal food products which could move in international trade without the threat of animal disease virus transmission.

STATUS OF FOOD VIROLOGY RESEARCH

In the United States, there are ongoing virological studies on drinking water and estuary waters, on oysters and other shellfish, on use of secondary sewage effluents for the irrigation of food crops, on development of detec-

TABLE 3. *Survival of foot-and-mouth disease virus in dairy products prepared from milk of infected cows.*

Product	Processing condition	Effect of processing on virus[a]	Reference
Whole milk	110°C/30 s	−	de Leeuw and Van Bekkum (*25*)
	148°C/2 s	+	Cunliffee, et al. (*23*)
Cream	93°C/15 s	−	Hyde et al. (*32*)
Cultured butter	93°C/15 s fermentation	--Minimum of 4 months	Blackwell (*6*)
Casein	72°C/0.25 minimum isoelectric ppt, pH 4.6	−42 d at 25°C	Cunliffe and Blackwell (*22*)
Cheese	No heat	−90 d +120 d curing	Blackwell (*5*)
Cheddar	63°C/10 s 67°C/15 s	−, processing, but not 30 d curing	
Camembert	72°C/15 s	−21 d +35 d curing	
Mozzarella	72°C/15 s	+processing alone	
Whey			
Acid	72°C/15 s pH 4.6	+processing alone	Blackwell (*7*)
Sweet	72°C/15 s pH 5.2		Blackwell (*7*)

[a] − Virus survived processing condition, + Virus inactivated by processing conditions.

tion methods, and on persistence of viruses during food processing. A large percentage of these studies emphasize water, because it is the principal carrier of viruses. The use of secondary sewage for irrigation of food crops should be discouraged, especially when the foods are to be consumed raw (*36*). Contamination of estuarian water by viruses may result in the bioaccumulation of viruses by the shellfish from the sea water. Unfortunately the viruses may persist in the shellfish for several days even after they have been transferred to clean water (*35*). Viruses with potential as food contaminants and their estimated status for laboratory studies are shown in Table 4.

The diversity and potential serverity of foodborne viral diseases of man or other animals are well established. It is also well established by the available evidence that we do not known or even have the means of establishing the true extent of foodborne viral diseases. The potential severity and economic impact of foodborne viral diseases supports the conclusion of a panel at the 1984 annual meeting of the American Society for Microbiology that additional research should be supported in government and academic laboratories to develop better methods for their detection, to determine the contribution of foodborne viruses to diseases of man, and to develop methods for prevention of foodborne viral diseases of man and animals.

REFERENCES

1. Anonymous. 1983. African swine fever. Italy. Bull. Off int. Epiz. 95:24-25.
2. Anonymous. 1960. Sur la survie du virus de la peste porcine classique dans la viande de porc et dans les produits de charcuterie. Documents sur la Persistance des Virus de Certaines Malades Animales dans les Viandes et Produits Animaux. O.I.E. p. 38-54.
3. Bachrach, H. L., S. S. Breese, J. J. Callis, W. F. Hess, and R. Patty. 1967. Inactivation of foot-and-mouth disease virus by pH and temperature changes and by formaldehyde. Proc. Soc. Exp. Biol. Med. 95:147-152.
4. Baldelli, B., A. Begliomini, T. Frescura, and D. Massa. 1965. Employment of gamma radiations for sterilizing the carcasses of foot-and-mouth disease virus infected animals. Soc. Ital. Sci. Vet. Atti. 19:538-542.
5. Blackwell, J. H. 1976. Survival of foot-and-mouth disease virus in cheese. J. Dairy Sci. 59:1574-1579.
6. Blackwell, J. H. 1978. Persistence of foot-and-mouth disease virus in butter and butter oil. J. Dairy Res. 45:283-285.
7. Blackwell, J. H. 1978. Potential transmission of foot-and-mouth disease in whey constituents. J. Food Prot. 41:631-633.
8. Blackwell, J. H. 1980. Internationalism and survival of foot-and-mouth disease virus in cattle and food products. J. Dairy Sci. 63:1019-1030.
9. Blackwell, J. H. 1984. Foreign animal disease agent survival in animal products: Recent developments. J. Am. Vet. Med. Assoc. 184:674-679.
10. Blackwell, J. H., and J. L. Hyde. 1976. Effect of heat on foot-and-mouth disease virus (FMDV) in the components of milk from FMDV-infected cows. J. Hyg. Camb. 77:77-83.
11. Blackwell, J. H., P. D. McKercher, F. V. Kosikowski, and R. G. Gorewit. 1982. Concentration of foot-and-mouth disease virus in milk of cows infected under simulated field conditions. J. Dairy Sci. 65:1624-1631.
12. Blackwell, J. H., P. D. McKercher, F. V. Kosikowski, and R. G. Gorewit. 1983. Physiochemical transformation of milk components and release of foot-and-mouth disease virus. J. Dairy Res. 50:17-25.
13. Blackwell, J. H., D. A. Rickansrud, P. D. McKercher, and J. W. McVicar. 1982. Effect of thermal processing on the survival of foot-and-mouth disease virus in ground meat. J. Food Sci. 47:388-392.
14. Blackwell, J. H., S. R. Wool, and F. V. Kosikowski. 1981. Vesicular exocytosis of foot-and-mouth disease virus from mammary gland secretory epithelium of infected cows. J. Gen. Virol. 56:207-212.
15. Botija, S. C. 1962. Studies on African Swine Fever in Spain. Int. Off. of Epizooties, XXXth Session.
16. Burrows, R. 1968. Excretion of foot-and-mouth disease virus prior to the development of lesions. Vet. Rec. 82:387-388.

TABLE 4. *Virology research priorities.*

Virus (human)	Grown in culture	Method of detection	Research potential[a]
Parvoviruses	Yes	EM, HA	10
Caliciviruses (Norwalk)	No	EM	1
Enteroviruses	Yes	Variety of methods	3
Polioviruses, Coxsackieviruses A and B Echoviruses	Variety of cultures needed, lab animals		
Hepatitis A (Enterovirus 72)	Yes Slow, cell associated, direct isolation quantitation questionable	EM, immunological methods	8
Rotaviruses	Yes Direct isolation quantitation not presently available	EM, immunological methods	8
Reoviruses	Yes	Variety of methods	2
Papoviruses	Yes Culture requirements difficult	Immunological methods	4
Astroviruses	No	EM	1

[a]Low potential -0; high potential -10.

17. Burrows, R., J. A. Mann, A. Greig, W. G. Chapman, and D. Goodridge. 1971. The growth and persistence of foot-and-mouth disease virus in the bovine mammary gland. J. Hyg. Camb. 69:307-321.
18. Cliver, D. O., R. D. Ellender, and M. D. Sobsey. 1983. Methods for detecting viruses in foods: Background and general principles. J. Food Prot. 46:248-259.
19. Cliver, D. O., R. D. Ellender, and M. D. Sobsey. 1983. Methods to detect viruses in foods: Testing and interpretation of results. J. Food Prot. 46:345-357.
20. Cottral, G. E. 1969. Persistence of foot-and-mouth disease virus in animals, their products and the environment. Bull. Off. Int. Epiz. 71:549-568.
21. Cottral, G. E., B. G. Cox, and D. E. Baldwin. 1960. The survival of foot-and-mouth disease virus in cured and uncured meat. Am. J. Vet. Res. 21:288-297.
22. Cunliffe, H. R., and J. H. Blackwell. 1977. Survival of foot-and-mouth disease virus in casein and sodium caseinate produced from milk of infected cows. J. Food Prot. 40:389-392.
23. Cunliffe, H. R., J. H. Blackwell, R. Dors, and J. S. Walker. 1979. Inactivation of milkborne foot-and-mouth disease virus at ultra-high temperature. J. Food Prot. 42:135-137.
24. Dawe, P. S. 1974. Viability of swine vesicular disease in carcasses and faeces. Vet. Rec. 94:430.
25. DeLeeuw, P. W., and J. G. VanBekkum. 1979. Report of the session of the Research Group of the Standing Technical Committee of the European Commission for the Control of Foot-and-Mouth disease. Lindholm, Denmark 12-14 June.
26. Dhennin, L., A. Frouin, B. Gicquel, J. P. Bidard, and J. Labie. 1980. Risk of disseminating foot-and-mouth disease virus by uncooked meat products. Bulletin de L'Adademie Veterinarie de France 53:315-322.
27. Dull, H. B., T. C. Doege, and J. W. Mosley. 1963. An outbreak of infectious hepatitis associated with a school cafeteria. Southern Med. J. 56:475-480.
28. Garcia-Vidal, W., C. Correa, H. Lazaneo, V. Urrestarazu, S. Huertas, and N. D. Heidelbaugh. 1984. Review of recent progress of the Meat Institute of Uruguay on the development of industrial methods to inactivate foot-and-mouth disease virus in meat and meat products. J. Vet. Res. (In press).
29. Gillespie, J. H., and J. F. Timoney. 1981. Rinderperst. Hagan and Bruner's infectious disease of domestic animals. Cornell University Press, Ithaca, NY. pp. 734-742.
30. Graves, J. H., and P. D. McKercher. 1975. Swine vesicular disease. pp. 346-352. *In* H. W. Dunne and A. D. Leaman, (ed.), Diseases of swine, 4th ed. Iowa State University Press. Ames, Iowa.
31. Heidelbaugh, N. D., and J. H. Graves. 1968. Effects of some techniques applicable in food processing on the infectivity of foot-and-mouth disease virus. Food Technol. 22:120-124.
32. Hyde, J. L., J. H. Blackwell, and J. J. Callis. 1975. Effect of pasteurization and evaporation on foot-and-mouth disease virus in whole milk from infected cows. Can. J. Comp. Med. 39:305-309.
33. Knight, C. A. 1975. Action of chemical and physical agents on viruses. Chemistry of viruses, 2nd ed. Springer-Verlag, New York, NY. pp. 180-238.
34. Kowalenko, J., M. Sidorow, and L. Burba. 1965. African swine fever and control measures. Internationale Zeitung der Landwirtschaft deutsche No. 1, pp. 47-52.
35. Larkin, E. P. 1981. Food contaminants-viruses. J. Food Prot. 44:320-325.
36. Larkin, E. P., J. T. Tierney, J. Lovett, D. Van Donsel, D. W. Francis, and G. J. Jackson. 1978. Land application of sewage wastes: Potential for contamination of foodstuffs and agricultural soils by viruses, bacterial pathogens and parasites. pp. 315-323. *In* H. L. McKim (Symp. Coord.), State of knowledge in land treatment of wastewater, Vol. 2, Int. Symp. Aug. 20-25, Hanover, NH.
37. Machado, Jr., M. A. 1969. AFTOSA. Historical survey of foot-and-mouth disease and inter-American relations. State University of New York Press, Albany, NY.
38. McCauley, E. H., J. C. New, W. H. Miller, and N. A. Aulagi. 1977. The potential economic impact of foot-and-mouth disease in United States: A study conducted by the University of Minnesota under a cooperative agreement with the Animal and Plant Health Inspection Service, U.S. Department of Agriculture.
39. McKercher, P. D., J. H. Graves, J. J. Callis, and F. Carmichael. 1974. Swine vesicular disease: Virus survival in pork products. Proceedings 78th Annu. Meet, U.S. Anim. Health Assoc. pp. 213a-213g.
40. McKercher, P. D., J. H. Graves, J. J. Callis, and F. Carmichael. 1975. Swine vesicular disease: viral survival in pork products. Proc. of the Session of the Res. Group of Standing Tech. Comm. Europ. Comm. for the Contract FMD Brescia, Italy, pp. 162-167.32.
41. McKercher, P. D., W. R. Hess, and F. Hamdy. 1978. Residual viruses in pork products. Appl. Environ. Microbiol. 35:142-145.
42. McKercher, P. D., D. O. Morgan, J. W. McVicar, and N. J. Shout. 1980. Thermal processing to inactivate viruses in meat products. Proceedings 84th Meeting. U.S. Animal Health Assoc., pp. 320-328.
43. McVicar, J. W., C. A. Mebus, A. Brynjolfsson, and J. S. Walker. 1982. Inactivation of African swine fever virus in tissues by gamma radiation. Am. J. Vet. Res. 43:318-319.
44. Preston, P. T. 1978. Animal disease and imported foods. Food Technol. Austral., pp. 426-429.
45. Sanchez, B. C. 1962. Desarrolo de planes de lucha contra la peste porcina africana. United Nations Conf. Appl. Sci. Technol. Benefit Less Developed Areas 11:517, p. 6.
46. Sellers, R. F. 1968. The inactivation of foot-and-mouth disease by chemicals and disinfectants. Vet. Rec. 83:504-506.
47. Stewart, W. C., and D. R. Downing. 1979. Thermal inactivation of hog cholera virus in ham. Am. J. Vet. Res. 40:739-741.
48. Studies on foot-and-mouth disease. 1966. U.S. Nat. Acad. Sci., Publication 1345.
49. United States Department of Agriculture. 1976. Code of Federal Regulations, Animal and Plant Health Inspection Service, Title 9, Animals and Animal Products, Sect. 94.12, p. 11. U.S. Department of Agriculture, Washington, DC.
50. U.S. Department of Health, Education, and Welfare. 1965. Reports of common vehicle epidemics. Hepatitis Surveillance, no. 24, Communicable Disease Center, Atlanta, GA, 12, 13:16-19.
51. U.S. Department of Health, Education, and Welfare. 1971. Infectious hepatitis - Tennessee. Morbidity Mortality Weekly Report 20:357.